to clinically observed differences in PV flow. *Methods:* A mathematical model of the atrium with two PVs was developed using an elastance model to describe pressure-volume relations. Using the unsteady Bernoulli equation in a system of differential equations, an analytical solution of systolic PV flow in each vein was achieved. Using the same conditions of initial atrial pressure (10 mmHg) and relaxation, PV length was varied in one vein (5–15 cm), while PV length remained constant (10 cm) in the other PV. *Results:* As PV length increased (more blood to be accelerated and greater inertia), peak systolic PV flow decreased from 105 to 50 cm/s in that vein, while peak flow increased in the other from 55 to 70 cm/s, even though its length was constant, reflecting the reduced LA pressure response to less volume influx from the other PV. *Conclusions:* PV inertance (length) is a major determinant of PV flow. As PV inertia increases, peak systolic flow velocity decreases. PV flow in one vein influences flow in another. Hence, differences in PV vein length and inertance may be responsible for the differences in PV flow seen clinically in the same atrium, and may also mimic or mask differences created by MR jets.

1021-59 Influence of Cardiac Pacing Mode on Left Atrial Appendage Function: Implication for the Cause of Systemic Embolism in VVI Pacing

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A higher incidence of systemic embolism has been observed in patients with VVI pacing compared to DDD pacing. However, the cause of systemic embolism in VVI pacing remains unclear. Previous studies have demonstrated that thrombus formation and systemic embolization are related to left atrial appendage (LAA) dysfunction. To investigate the influence of pacing mode on LAA function, we performed transesophageal echocardiography in 16 patients (mean age 65) with permanent DDD pacemakers. The indications for pacemaker implantation were third degree atrio-ventricular block (A-V block, n = 10) and sick sinus syndrome (SSS, n = 6). All had shown sinus rhythm at implantation. We measured peak LAA emptying and filling flow velocities during both DDD and VVI pacing at 60 beats/min. The data are summarized below:

	Peak LAA flow velocity (cm/s)		p value
	DDD	VVI	
A-V block (n = 10)	58 ± 18	40 ± 19	<0.01
SSS (n = 6)	41 ± 13	24 ± 7	<0.05
Total (n = 16)	52 ± 18	34 ± 18	<0.01

Peak LAA flow velocities decreased significantly when a pacemaker was reprogrammed from DDD to VVI pacing mode, especially in patients with SSS.

Conclusion: LAA function was impaired in VVI pacing compared with that in DDD pacing. These results suggest that impaired LAA function in VVI pacing may predispose the chamber to thrombus formation, which may play a role in the mechanism of the embolization.

1021-60 Patients Presenting with Systemic Emboli are Often Found to Have Mobile Plaque in the Aortic Arch by Transesophageal Echocardiography (TEE)

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Although the mobile component of plaque is thought to represent thrombus, the efficacy of anticoagulation has not been proven. In order to assess the influence of anticoagulation in this population, we studied 29 pts with mobile plaques found by TEE interrogation of the aortic arch. Each pt had had a recent systemic embolism; cerebral in 27 (93%) and peripheral in 2 (7%). After TEE, pt treatment was determined by physician preference. Follow-up (mean 13 ± 12 mo.s) was obtained via telephone interview and chart review. Of the 29 pts studied, 19 (66%) received warfarin (WAR) and 10 (34%) received either aspirin (7) or no therapy(3). These groups were compared in regards to dimensions of both mobile and immobile plaque components and vascular events:

	Plaque Dimensions						Vascular Event
	Immobile Component			Mobile Component			
	Height	Width	Area	Height	Width	Area	
WAR (n = 19)	7 ± 3	29 ± 13	13 ± 6	8 ± 9	3 ± 3	5 ± 8	0
No WAR (n = 10)	6 ± 2	30 ± 15	12 ± 8	7 ± 4	3 ± 2	2 ± 3	6 (60%)
P	NS	NS	NS	NS	NS	NS	0.0009

The dimensions of both mobile and immobile components of plaque were similar in the groups. Vascular events occurred in 6 (60%) of the No WAR group (4 strokes and 2 myocardial infarctions) while none of the pts receiving warfarin had an event (p = 0.0009). Similarly when pts with and without vascular events were compared, no differences in plaque dimensions were found. In conclusion, mobile aortic plaque is associated with a high frequency of vascular events during a relatively brief period of follow-up. Although no morphologic aspect of the plaque predicts an event, it appears that warfarin may provide adequate prophylaxis.

1022 Clinical Studies in AMI

Wednesday, March 22, 1995, 3:00 p.m.–5:00 p.m.
Ernest N. Morial Convention Center, Hall E
Presentation Hour: 4:00 p.m.–5:00 p.m.

1022-101 Converting Enzyme Inhibition Decreases Infarct Collagen and Limits Hypertrophy of Non-Infarct Myocardium During Healing After Infarction

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Inhibition of tissue angiotensin-converting-enzyme (ACE) activity can potentially block both myocyte and fibroblast growth. The effects of 6 weeks of ACE inhibition with enalapril (2.5 mg b.i.d.) and captopril (50 mg b.i.d.) on in vivo changes in LV mass (two-dimensional echocardiography) and post-mortem myocardial collagen content (hydroxyproline, mg/g dry weight) were measured during healing after anterior infarction (left anterior coronary artery ligation) in 63 instrumented dogs (34 ACE inhibition; 29 placebo). Compared to the placebo group, both ACE inhibitors prevented the increase in LV diastolic volume (+51% versus −1.5%, p < 0.001) and LV mass (−3% versus +13%, p < 0.005) over the 6 weeks. However, compared to placebo, ACE inhibition decreased infarct collagen but not non-infarct collagen.

	Captopril (23)	Enalapril (11)	Placebo (29)
Infarct center	31 ± 2*†	23 ± 5*	45 ± 3
Normal zone	5 ± 0	5 ± 0	5 ± 0

*p < 0.001 vs. placebo, †p < 0.1, captopril vs. enalapril

Thus, both enalapril and captopril prevented LV dilatation and hypertrophy during postinfarct healing, but enalapril decreased infarct collagen slightly more than captopril. Differences in the effects of ACE inhibitors on myocyte and fibroblast growth may explain some differences in their effects on postinfarct remodeling, especially with large infarctions.

1022-102 Mortality Results of the Trandolapril Cardiac Evaluation (TRACE) Trial

Lars Køber, TRACE Study Group. *TRACE study office, Copenhagen, Denmark*

The aim of the TRAndolapril Cardiac Evaluation (TRACE) trial was to evaluate whether mortality of patients with left ventricular (LV) dysfunction shortly after myocardial infarction (MI) is reduced by long term treatment with an ACE-inhibitor, regardless of the presence of heart failure or ischemia.

7001 consecutive enzyme confirmed MIs in 27 Danish hospitals were screened for entry into the study 2–6 days following MI. At screening 2-dimensional echocardiography was recorded on videotape at the center and evaluated centrally. Wall motion index (WMI) was evaluated, and patients with WMI ≤ 1.2 (corresponding to left ventricular ejection fraction ≤ 0.35) were eligible for the study. Heart failure and ischemia did not exclude patients. After receiving a test dose, patients were randomly allocated to receive oral treatment with Trandolapril or placebo 3–7 days following the MI. The primary endpoint was all cause mortality (intention to treat). Follow up was a minimum of 2 years (range 2–4 years).

Results: 2606 (37%) of the screened MIs had WMI ≤ 1.2. This represented a high risk group with an overall 1-year mortality of 34%. In contrast, patients with WMI > 1.2 had a one year mortality of only 12%. Of patients eligible for the study based on LV function, 1749 patients (67%) were included.

876 patients received Trandolapril and 873 patients Placebo. Baseline characteristics did not differ. Trandolapril caused a highly significant reduction in overall mortality. Overall risk reduction was 22%, p = 0.0007. At study closure mortality of Trandolapril treated patients was 35% and that of placebo treated patients 42%.

Conclusion: Long term treatment with Trandolapril of patients with LV dysfunction shortly after MI, significantly reduced mortality. The study adds to the results of the SAVE trial by including a large proportion of eligible patients and not excluding patients with heart failure or ischemia.